

DEVELOPMENT OF AN INTELLIGENT STRETCHING DEVICE FOR ANKLE JOINTS WITH CONTRACTURE/SPASTICITY

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Abstract- A stretching device with intelligent control was developed to treat spastic/contractured ankles of neurologically impaired patients and evaluate the outcome. The device stretched the ankle joint safely throughout the range of motion (ROM) to extreme positions until a specified peak resistance torque was reached with the stretching velocity controlled based on the resistance torque. The ankle was then held at the extreme position for a period of time to let stress relax before it was rotated back to the other extreme position. The stretching was slow at the joint extreme positions, making it possible to reach a larger ROM safely, and it was fast in the middle ROM so the majority of the treatment was spent in stretching the problematic extreme ROM. The device was evaluated in five healthy subjects and used to treat four stroke patients. Furthermore, it was used to evaluate treatment outcome in multiple aspects, including passive/active joint ROM, stiffness, viscous damping, and reflex excitability. The intelligent control and yet simple design of the device suggest that the device can be made portable at relatively low cost, making it available to patients/therapists for frequent use in clinics/home and allowing more effective treatment and long-term improvement.

Keywords - stretch, stiffness, reflex, stroke

I. INTRODUCTION

Contracture and spasticity are major sources of disability in neurological impairment including stroke, spinal cord injury, multiple sclerosis, and cerebral palsy [1, 2]. There is a strong need to regularly treat and exercise joints of neurologically impaired patients effectively to reduce contracture and/or spasticity.

Manual stretching is important and effective in treating joints with limited mobility, spasticity and/or contracture [3, 4]. However, the effects may not be long lasting, partly due to the limited and sometime infrequent or insufficient therapy.

Several of methods have been developed to exercise the joint and reduce joint spasticity/contractures such as, serial casting, dynamic splinting and traction, the continuous passive motion (CPM) device, and advanced robot-aided devices [3-6]. However, existing devices like the CPM machine move the limb at a constant speed between two preset joint positions. When it is set within the flexible part of the ROM, the passive movement does not usually stretch into the extreme positions where contracture/spasticity is significant. On the other hand, setting a CPM machine too aggressively may risk injuring the joint. There is a need for a device that can safely stretch the joint to its extreme

positions with quantitative control of the resistance torque and stretching velocity. Furthermore, there is a strong need for quantitative and objective measures of the impairment and rehabilitation outcome.

The objectives of this study were 1) to develop a stretching device with intelligent control to stretch ankle joints with spasticity/contracture safely and repeatedly throughout the ankle ROM.; 2) to assess the feasibility of the stretching device on a small sample of neurologically impaired patients with ankle spasticity and/or contracture and to develop means of evaluating treatment outcome quantitatively in multiple aspects.

II. METHODOLOGY

1) *Subjects*: Five healthy subjects with no prior history of neurological disorders and neuromuscular injury were used to test the device and establish the protocol for the stretching treatment and outcome evaluation. Four stroke patients with ankle contracture and/or spasticity participated in the study.

2) *Experimental Setup*: Both the stretching and the outcome evaluation were done using a custom-designed joint stretching device (Fig. 1). The subject was seated with the thigh and trunk strapped to the adjustable seat and backrest, respectively. The leg was strapped to the leg support at 60° knee flexion and the hip flexed at ~85°. The foot was cast with fiberglass tape and coupled to a foot attachment through two half-rings. The foot-attachment was fixed to the motor shaft through a six-axis force sensor that measured the moments at the ankle joint. The foot attachment can be adjusted to align the ankle flexion axis with the motor shaft.

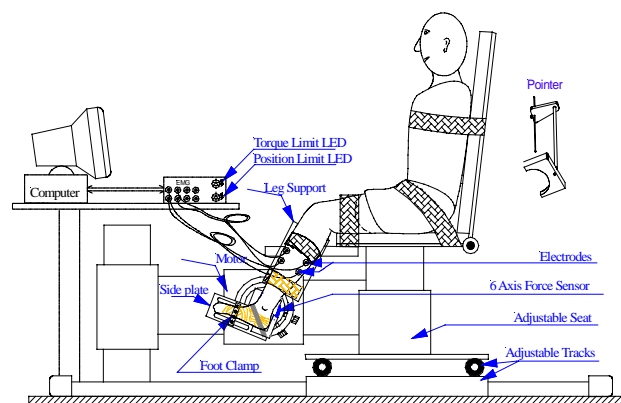


Fig. 1. Experimental setup for stretching the ankle joint and evaluating treatment outcome.

3) *Control of the Stretching*: The stretching device was driven by a servomotor controlled by a digital signal

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processor (DSP) controller (Fig. 1). A velocity-control loop was implemented by the DSP controller that read the joint position and resistance torque and controlled the stretching velocity accordingly. The stretching velocity was controlled in such a way that it was inversely proportional to the resistance torque. Near the end of ROM, the increasing resistance slowed down the motor gradually and the muscle-tendons involved were stretched slowly and safely. Once the specified peak resistance torque was reached, the motor held the joint at the extreme position for a period of time (e.g., 10 seconds, as is usually used by a physical therapist) that could be adjusted conveniently. In the middle ROM where the resistance was usually low, the motor stretched the slack muscles at higher speeds. As a safety precaution, position limits were set by the operator and they were monitored by the DSP controller. Mechanical and electrical stops were also used to restrict the motor range of motion. In addition, the operator and the subject each had a stop switch, and either of them could shut down the motor by pressing the switch.

4) *Evaluation of Intrinsic Mechanical Changes Caused by Stretching*: Measurement of ROM was done under the same peak resistance torque were compared between trials and between different subjects.

Quasi-static stiffness was measured as the slope of the torque-angle relationship (hysteresis loop) [7, 8]. The dorsi-flexion half of the hysteresis loop had a stretching (curve AB) and retreating (BCD) phases (Fig. 2). The slopes of the initial (line AA') and terminal (line B'B) segments of the stretching phase were characterized as the initial dorsi-flexion stiffness and terminal dorsi-flexion stiffness, respectively (Fig. 2). The initial plantar flexion stiffness and terminal plantar flexion stiffness were similarly defined.

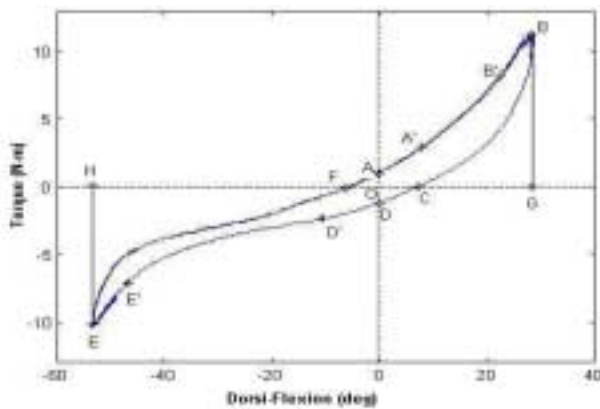


Fig. 2. Torque-angle relationship (hysteresis loop) during ankle stretching in dorsi- and plantar-flexion, obtained from a healthy subject. The x-axis is the dorsi-flexion angle and the Y-axis is the external dorsi-flexion torque or plantar flexor resistance torque.

Active and passive joint dynamic properties in terms of joint stiffness and viscosity before/after stretching were evaluated quantitatively. The stretching device applied small-amplitude random perturbations to the ankle while the subject either relaxed or maintained a steady level of plantar or dorsi-flexion torque throughout a trial. Joint stiffness, viscosity, and limb inertia were identified from the

measured joint position and torque based on system identification.

5) *Evaluation of Reflex Changes Caused by Stretching*: Reflex properties were evaluated quantitatively by tapping the Achilles tendon with an instrumented reflex hammer and measuring the reflex-mediated EMG and joint torque responses using the stretching device [9, 10]. The tendon tapping was done at isometric condition with the stretching device locking the ankle at the neutral position.

6) *Protocol*: At the beginning of the experiment, the subject was examined for spasticity and contracture using the Ashworth scale (0 to 4), tendon reflex scale (0 to 4), and joint range of motion.

Biomechanical and reflex properties of the ankle were then evaluated before the stretching treatment. First, with the ankle fixed at the neutral position by the device, the subject was asked to generate maximum voluntary contraction (MVC) in plantar flexion and in dorsi-flexion. The MVC evaluation was repeated twice and the maximum torques reached in plantar- and dorsi-flexion were taken. Second, intrinsic properties were manifested and evaluated using small-amplitude random perturbations with the subject maintaining a steady level of plantar- or dorsi-flexion torque throughout a trial, ranging from about -40% to 40% of the plantar-flexion torque, at the increment of 10% plantar-flexion torque. The negative percentage corresponded to background dorsi-flexion muscle contraction and 0% was the relaxed state.

Next, tendon reflexes were evaluated quantitatively by tapping the Achilles tendon with an instrumented reflex hammer and measuring the reflex responses.

Before stretching, the joint stretching device was rotated manually to the extreme dorsi-flexion and plantar flexion to set the extreme position limits (θ_p and θ_n). After the operator chose the M_p , M_n and θ_d values, the stretching device flexed the ankle throughout its ROM, with the DSP controller controlling the stretching velocity based on the resistance torque. The patient was asked to relax and not to react to the stretch. The maximum stretching velocity (typical value: 30°/sec, usually for the middle ROM with near-zero resistance), peak resistance torque, and length of the holding period at the joint extreme positions were specified.

After the stretching treatment, the above biomechanical and reflex evaluations were repeated to assess stretching-induced changes in the relevant properties. During the stretching treatment and the pre- and post-stretching evaluations, ankle flexion angle, joint torque, and EMG signals from the soleus, gastrocnemius and tibialis anterior muscles were recorded. All signals were sampled at 500 Hz after lowpass filtering with 230 Hz cutoff frequency. Active surface electrodes were used to record the EMG signals.

III. RESULTS

A. General Features of the Stretching Treatment

The well-controlled stretching was safe to the subjects and the device was easy to use for the operator/therapist. Fig. 3

shows a representative stretching trial. Subjectively, both healthy subjects and patients with neurological impairment liked the strenuous stretching, which made them feel like having a good workout on the ankle.

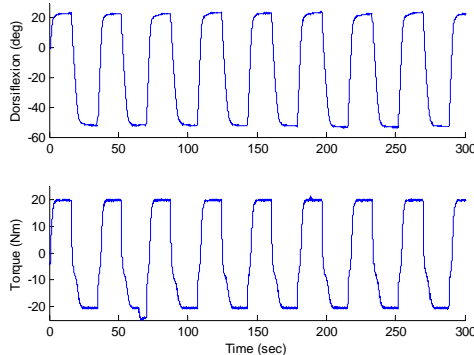


Fig. 3. Representative signals during an ankle-stretching treatment trial on a stroke patient with a spastic ankle. The top and bottom plots correspond to the dorsiflexion angle and external dorsiflexion torque, respectively. The ankle was stretched to both of the extreme positions and the stretching velocity was inversely proportional to the resistance torque.

B. Torque-Angle Relationship, Passive ROM and Changes Induced by Stretching

The relationship between the ankle dorsiflexion and external dorsiflexion torque (plantar flexor muscle resistance torque) during the strenuous stretching was quite different between healthy subjects and patients (Fig. 4). The ankle ROM in stroke was reduced markedly in both dorsiflexion and plantar flexion under comparable levels of torque. The dorsiflexion ROM for the healthy and stroke patient in Fig. 4 was 31.2° and 18.7° , respectively, at 20 N·m terminal torque. The plantar flexion ROM at 15 N·m terminal torque for the healthy and stroke patient was 58.1° and 50.0° , respectively.

The joint resistance to dorsiflexion movement rose much more quickly in the spastic ankle of the stroke patient than that in the normal one (Fig. 4). The resistance in plantar flexion in the spastic ankle of the stroke patient rose more quickly with plantar flexion than that in the healthy subject.

However, the quasi-static stiffness in the terminal zone was lower than that of the normal subject. The quasi-static stiffness in dorsiflexion was much higher than its counterpart in plantar flexion in a stroke patient with spastic ankle joint (the thin solid line in Fig. 4). The 1st round stretching showed stiffer joint than that in the 2nd round.

The passive ROM of the ankle joint increased considerably after the stretching treatment. For a representative case, dorsiflexion range increased from 11.9° to 16.5° at the same level of terminal torque (10 N·m) after a stretching session of 30 min. Similarly, plantar flexion range increased from 32.1° to 35.5° at 10 N·m torque. Over multiple stroke patients, the increase in ankle ROM was consistently observed in both dorsiflexion and plantar flexion, making the passive ROM closer to that of the healthy subjects.

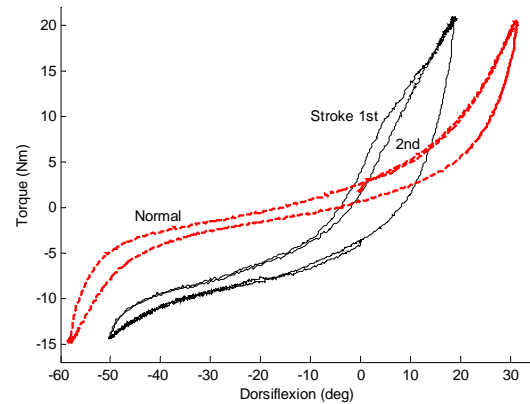


Fig. 4. The relationship between the ankle dorsiflexion and external dorsiflexion torque in a stroke patient with rigid ankle (the thin solid line) and in a normal subject (the thick dashed line). Notice the much higher stiffness (the steeper slope) in dorsiflexion caused by the spastic/contractured plantar flexors in the stroke patient and the considerable energy loss during the passive stretching of the plantar flexors. The curve moves clock-wise as time and the stretching progresses.

C. Changes in Active ROM Induced by Stretching

The strenuous stretching loosened the spastic joint of neurologically impaired patients. As a result, patients were able to move the joint voluntarily in a much larger range of motion. In a stroke patient with foot drop and wearing ankle-foot-orthosis, the spastic ankle was stretched forcefully to 20 N·m in extreme dorsiflexion and 15 N·m in extreme plantar flexion for 30 minutes. Before the stretching treatment, the active ROM was from 8.8° to 21.7° (both in plantar flexion because the subject could not reach the neutral position). After stretching, the active ROM increased to from 2.0° plantar flexion to 26.9° plantar flexion.

D. Changes in Joint Dynamic Control Properties Induced by Stretching

The stretching treatment with intelligent control resulted in considerable changes in joint dynamic control properties characterized by joint stiffness and viscous damping (Fig. 5). As shown in the representative cases, joint stiffness was reduced markedly after stretching across the range of muscle contraction (Fig. 5), including both passive (muscle relaxed) and active (muscle contracting) conditions. Similarly, the stretching treatment reduced the joint viscosity for both passive (muscles relaxed) and active ankle joint-muscles with muscles contracting at various levels (Fig. 5).

E. Changes in Reflex Properties Induced by Stretching

Since the stretching treatment loosened the spastic muscle-tendons and increased the joint ROM considerably in the patients, the Triceps Surae muscle and Achilles tendon became slacker and the reflex-mediated plantar flexor torque became much weaker after stretching (Fig. 6). Quantitatively, tendon reflex gain [9, 10] was reduced from 11.6 to 2.7 (cm). Strong clonus was observed at the ankle before stretching, which disappeared after stretching, possibly related to the slacker state of the Triceps Surae.

muscle and Achilles tendon. Similar reductions in reflex excitability after the stretching exercise were observed in the other patients as well.

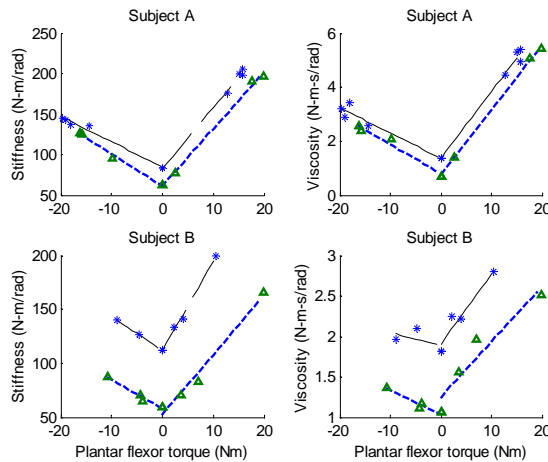


Fig. 5. Changes in joint stiffness K and viscosity B caused by the stretching treatment of spastic ankles in two stroke patients. The “*” and “ Δ ” symbols correspond evaluations done immediately before and after stretching, respectively. The variables are characterized as functions of muscle torque with the positive torque corresponding to plantar flexor contraction.

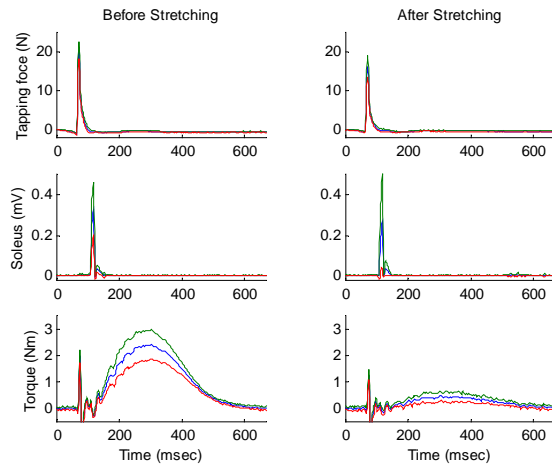


Fig. 6. Tendon tapping results over multiple taps on the Achilles tendon of a stroke patient with spastic ankle. The solid and dashed lines give the mean and $\text{mean} \pm \sigma$ of each signal, respectively. The initial spike in the reflex torque signal (at about 80ms) was from the mechanical vibration caused by the taps.

IV. DISCUSSION

The study presented a useful method to treat the spastic/contractured ankles of neurologically impaired patients and evaluate the treatment outcome in multiple aspects, including active and passive ROM, joint stiffness and viscous damping, and reflex excitability. The intelligent and forceful stretching reduced both joint stiffness and viscosity in the spastic ankle of stroke patients. Reflex-mediated joint torque response was reduced substantially after the stretching treatment.

A limitation of the study was that only short-term improvements in ankle flexibility, and joint intrinsic and reflex properties were shown. Another limitation is that the above stretching device is large and not portable. Further study is being carried out to study the long-term effect of the stretching treatment. The intelligent stretching device can be made portable, which will make it easier for patients and therapists to use it at home or in clinics.

V. CONCLUSION

This study shows that the intelligently controlled stretching device can stretch the ankle forcefully and safely to its extreme positions, resulting in considerable improvement of treatment outcome in the sample of spastic patients, which was evaluated in several aspects. The simple design of the device suggests that the device can be made portable with relatively low cost, making it available to patients and therapists for frequent use in clinics/home and allowing more effective treatment and long-term improvement.

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